L2 ANSWER 8 OF 11 USPATFULL

SUMM Baggiolini et al. in European Patent Publication No. 580,968 disclose fluorinated vitamin D.sub.3 analogs, including 1.alpha.-fluoro-25-hydroxy-16-ene-23-yne-26,27-hexafluorochole-calciferol, useful for the treatment of hyperproliferative disorders of the skin, for the treatment of cancer and leukemia, and for the treatment of **sebaceous gland diseases**. U.S. patent application Ser. No. 08/560,080 discloses and claims the use of this compound for the restoration of bone mass and/or density in osteoporosis. The disclosures of the cited references are hereby incorporated by reference.

ACCESSION NUMBER:	1999:22094 USPATFULL			
TITLE:	Fluorinated vitamin D3 analogs			
INVENTOR(S):	Nestor, Jr., John J., Louisville, KY, United States Manchand, Percy S., Montclair, NJ, United States Uskokovic, Milan R., Upper Montclair, NJ, United States Vickery, Brian H., Los Altos Hills, CA, United States			
PATENT ASSIGNEE(S):	Syntex (U.S.A.) Inc., Palo Alto, CA, United States (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5872113		19990216	
APPLICATION INFO.:	US 1997-857569		19970516	(8)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Lambkin, Deborah C.			
LEGAL REPRESENTATIVE:	Peries, Rohan			
NUMBER OF CLAIMS:	27			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	43 Drawing Figure(s); 43 Drawing Page(s)			
LINE COUNT:	1643			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L2 ANSWER 9 OF 11 USPATFULL

SUMM Baggiolini et al. in European Patent Publication No. 580,968 disclose fluorinated vitamin D.sub.3 analogs, including 1.alpha.-fluoro-25-hydroxy-16-ene-23-yne-26,27-hexafluorocholecalciferol, useful for the treatment of hyperproliferative disorders of the skin, for the treatment of cancer and leukemia, and for the treatment of **sebaceous gland diseases**. Use for the restoration of bone mass and/or density in osteoporosis is not suggested.

ACCESSION NUMBER:	97:115262 USPATFULL			
TITLE:	Method for treating osteoporosis			
INVENTOR(S):	Nestor, Jr., John Joseph, Cupertino, CA, United States Vickery, Brian Henry, Mountain View, CA, United States Uskokovic, Milan Radoje, Upper Montclair, NJ, United States			
PATENT ASSIGNEE(S):	Syntex (U.S.A.) Inc., Palo Alto, CA, United States (U.S. corporation) Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5696103		19971209	
APPLICATION INFO.:	US 1995-560080		19951117	(8)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Ivy, C. Warren			
ASSISTANT EXAMINER:	Mach, D. Margaret M.			
LEGAL REPRESENTATIVE:	Heller Ehrman White & McAuliffe			

L2 ANSWER 11 OF 11 USPATFULL

SUMM It is also a primary object of the invention to provide a composition which utilizes the cation exchange capacity of certain clay minerals to release and deliver ~~magnesium cations~~ (Mg.sup.++) and hydroxyl anions (OH.sup.31) to diseased dermal growth cells and **diseased sebaceous gland** cells to effectively treat epidermal and sebaceous disorder.

ACCESSION NUMBER:	90:44341 USPATFULL		
TITLE:	Composition for the effective treatment of scalp diseases that delivers magnesium adsorbed in alumina silicate clays to affected sites		
INVENTOR(S):	Barabino, William A., North Reading, MA, United States Cross, Robert J., Haverhill, MA, United States		
PATENT ASSIGNEE(S):	Physiological Research Associates, North Reading, MA, United States (U.S. corporation)		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 4931274		19900605
APPLICATION INFO.:	US 1988-233033		19880817 (7)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1986-835443, filed on 27 Feb 1986, now abandoned which is a continuation of Ser. No. US 1984-606975, filed on 4 May 1984, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Ore, Dale R.		
LEGAL REPRESENTATIVE:	Litman, Richard C.		
NUMBER OF CLAIMS:	16		
EXEMPLARY CLAIM:	1		
LINE COUNT:	725		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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DETD [0149] Liver Cancer **Photodynamic** Therapy by Transillumination
DETD [0152] Rapid Tissue Clearance and Prolonged Tumor Retention followed by Transcutaneous **Photodynamic** Therapy
DETD [0157] Additionally, the quantum mechanics of transcutaneous **photodynamic** therapy result in an amplification of the photosensitizer agent. Since each molecule of the photosensitizer agent is repeatedly activated upon transcutaneous illumination, a relatively low dose of the photosensitizer agent can be highly effective in destroying tumor tissue. Whether through singlet oxygen production upon photoactivation or stimulation of an immune response or both, transcutaneous **photodynamic** therapy demonstrates less adverse reaction or collateral normal tissue damage than most other forms of cancer therapy.
DETD [0171] Exceeding the **photodynamic** threshold using extended low light level PDT is tumoricidal.
CLM What is claimed is:
9. The method of claim 7, wherein said photosensitizer compound is selected from the group consisting of indocyanine green, **methylene blue**, toluidine blue, **aminolevulinic acid**, chlorins, phthalocyanines, porphyrins, purpurins, and texaphyrins.

ACCESSION NUMBER: 2002:165569 USPATFULL
TITLE: Transcutaneous **photodynamic** treatment of targeted cells
INVENTOR(S): Chen, James, Bellevue, WA, UNITED STATES
PATENT ASSIGNEE(S): Light Sciences Corporation (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002087205	A1	20020704
APPLICATION INFO.:	US 2001-905501	A1	20010713 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. WO 2000-US944, filed on 14 Jan 2000, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-116234P	19990115 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Stephanie L. Seidman, Heller Ehrman White & McAuliffe, LLP, 4350 La Jolla Village Drive, 6th Floor, San Diego, CA, 92122-1246
NUMBER OF CLAIMS: 30
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Page(s)
LINE COUNT: 1774

TFULL on STN

TI Inhibition of fibrosis by **photodynamic** therapy

AB Disclosed is a method for modulating wound healing in a mammal. The method includes the steps of: (a) administering a photosensitizer to a mammal that has an unhealed or partially-healed wound; (b) waiting for the photosensitizer to reach an effective tissue concentration at the wound site; (c) photoactivating the photosensitizer by delivering specifically to the wound site light of a effective wavelength and intensity, for an effective length of time. The modulation of wound healing can include hastening healing by administering a low dose of **photodynamic** therapy. Alternatively, the modulation can include inhibiting fibrosis by administering a high dose of **photodynamic** therapy. The photosensitizer can be targeted, for example, to macrophages or myofibroblasts. Targeting can be by conjugation to a targeting moiety such as a protein, peptide or microparticle.

SUMM This invention relates to wound healing and **photodynamic** therapy.

SUMM Macrophages are central to the complex process of wound healing, which involves removal of dead tissue, formulation of granulation tissue, neovascularization, stimulation of locomotion and proliferation of fibroblasts and keratinocytes, and production of collagen types I and III. **Photodynamic** therapy can destroy large amounts of tissue with a good healing response and good cosmetic result (Koren et al, Int. J. Radiat. Oncol. Biol. Phys. 28:463-466 (1994)). **Photodynamic** therapy can be used either to stimulate or suppress cellular responses such as cytokine release and immune function. Whether the **photodynamic** therapy causes stimulation or suppresion depends on the dosage. Low dose **photodynamic** therapy stimulates cytokine release and immune function, while high dose **photodynamic** therapy suppresses those processes (Obochi et al., SPIE Proc. 2675:122-131 (1996); Yamamoto et al., Photochem. Photobiol. 60:160-164 (1994)).

SUMM **Photodynamic** therapy has major effects on macrophages. Low dose **photodynamic** therapy activates macrophages. This enhances their cytotoxicity against tumor cells (Yamamoto et al., Photobiol. B 13:295-306 (1992)). High dose **photodynamic** therapy leads to production of TNF alpha, and eventually, macrophage death (Evans et al., J. Natl. Cancer Inst. 82:34-39 (1990)).

SUMM **Photodynamic** therapy has been used to treat cancer. See, e.g., Dougherty et al., In **Photodynamic** Therapy of Neoplastic Disease, (Kessel, ed.), CRC Press, Boca Raton, Fla. (1989). **Photodynamic** therapy has also been used for destruction of the synovium in the treatment of rheumatoid arthritis (U.S. Pat. No. 5,368,841).

SUMM The invention features a method for modulating the healing of a wound in a mammal. The method includes the steps of: (a) administering an effective amount of a photosensitizer to a mammal that has an unhealed or partially-healed wound; (b) waiting for a time period wherein the photosensitizer reaches an effective tissue concentration at the wound site; (c) photoactivating the photosensitizer at the wound site by delivering specifically to the wound site light of a effective wavelength and intensity, for an effective length of time. The modulation of wound healing can include hastening healing by administering a low dose of **photodynamic** therapy. Alternatively, the modulation can include inhibiting fibrosis by administering a high dose of **photodynamic** therapy.

SUMM Photosensitizers include members of the following classes of compounds: porphyrins, chlorins, bacteriochlorins, purpurins, phthalocyanines, naphthalocyanines, texaphyrins, and non-tetrapyrrole photosensitizers.

Specific examples include Photofrin, benzoporphyrin derivative, tin etiopurpurin, sulfonated chloroaluminum phthalocyanine and **methylene blue**. The photosensitizer can be targeted, for example, to macrophages or myofibroblasts, by conjugation to a targeting moiety such as a protein, peptide, or microparticle. Administration of the photosensitizer can be local or systemic. For systemic administration, the preferred dosage is between about 0.1 mg/kg and about 50 mg/kg. More preferably, it is at a dosage level between about 0.5 mg/kg and about 10 mg/kg. In other embodiments of the invention, the administration of the photosensitizer is local. Local administration can be perilesional or topical.

SUMM As used herein, "low dose" **photodynamic** therapy means a dose sufficient to kill from 0% to about 10% of all cells exposed to the photoactiviting light if the photosensitizer is untargeted, or from 0% to about 10% of the targeted cells exposed to the photoactiviting light, if the photosensitizer is targeted. As used herein, "high dose" **photodynamic** therapy means a dose sufficient to kill from about 10% to about 90% of all cells exposed to the photoactivating light if the photosensitizer is untargeted, or from about 10% to about 90% of the targeted cells exposed to the photoactivating light, if the photosensitizer is targeted.

SUMM As used herein, "high dose" **photodynamic** therapy means a dose sufficient to kill from about 30% to about 100% of all cells exposed to the photoactivating light if the photosensitizer is untargeted, or from about 30% to about 100% of the targeted cells exposed to the photoactivating light, if the photosensitizer is targeted. The dose of **photodynamic** therapy is calculated as the product of photosensitizer dose and photoactivating light dose. Thus, **photodynamic** therapy dose can be adjusted by adjusting the photosensitizer dose, photoactivating light dose, or both.

DETD The present invention involves applying **photodynamic** therapy ("PDT") to an unhealed or partially healed wound. In general, **photodynamic** therapy involves administration of a photosensitizer to a patient, followed by photoactivation of the photosensitizer, to produce a cytotoxic effect. In the present invention, photoactivating light is delivered specifically to an unhealed or partially-healed wound, where the biological effect modulates wound healing.

DETD The modulation of wound healing can be achieved according to this invention by modulating macrophage function, myofibroblast function, endothelial cell function, or any combination thereof, through **photodynamic** therapy at a wound site. Preferaby, the **photodynamic** therapy includes targeting a photosensitizer to macrophages and myofibroblasts. Targeting can be accomplished, for example, by conjugating the photosensitizer to a targeting moiety that binds to a receptor on the macrophage or myofibroblast surface, e.g., an LDL receptor or a "scavenger" receptor. Alternatively, macrophage targeting can be accomplished by exploiting the phagocytosis that characterizes macrophages and myofibroblasts. The photosensitizer can be conjugated to a microparticle, e.g., a 1 μm polystyrene microsphere (Polysciences, Inc.). Such photosensitizer-microparticle conjugates are taken up selectively by macrophages and myofibroblasts, due to phagocytotic activity of those cell types. A photosensitizer-microparticle conjugate can be produced by known methods, e.g., those described in Bachor et al., Proc. Natl. Acad. Sci. USA 88:1580-1584 (1991).

DETD Photosensitizers include members of the following classes of compounds: porphyrins, chlorins, bacteriochlorins, purpurins, phthalocyanines, naphthalocyanines, texaphyrines, and non-tetrapyrrole photosensitizers. Specific examples are Photofrin, benzoporphyrin derivative, tin etiopurpurin, sulfonated chloroaluminum phthalocyanine and

methylene blue. BPD is a second generation porphyrin photosensitizer that diffuses rapidly from microvasculature and disseminates throughout a joint. In addition, BPD has a low affinity for chondrocytes and articular cartilage following systemic or intra-articular injection. CASPc, a phthalocyanine inactivates growth factors TGF-β and bFGF.

DETD An alternative to administration of the photosensitizer compound itself, is administration of a photosensitizer precursor molecule. This approach is illustrated by the use of 5-**aminolevulinic acid**, which causes endogenous production of the photosensitizer protoporphyrin IX (Morgan et al., J. Med. Chem. 32:904-908 (1989).

DETD Macrophage Targeted **Photodynamic** Regulation of Wound Healing

DETD In this protocol, 64 hairless rats receive multiple surgical incisions to their backs. Incisions are applied at 2 time points prior to, and 1 time point after, application of **photodynamic** therapy. The **photodynamic** therapy entail systemic or local photosensitizer administration followed by regional light therapy.

DETD At 3 days prior to, one hour prior to, and one hour after **photodynamic** treatment, a series of full thickness incisions are made with a #10 scalpel blade on the back of each animal. Incisions are 25 mm in length, and spaced 1 inch apart. At time 0, rats 1-24 receive systemic injection of varying concentrations (0.5 mg/kg to 10 mg/kg) of CASP via a 30 gauge needle into the tail vein. Wounds are irradiated with 25, 50 or 100J laser energy at a wavelength of 675 nm at 5, 180 minutes, and 24 hours post injection. Animals 25-48 are treated with topical application of **methylene blue** 5 minutes before, and one hour before, photo activation. Using similar laser treatment parameters, 660 nm light is applied to the incision sites. Animals 49-56 are treated with systemic injections of BPD-MA (0.5 mg/kg to 10 mg/kg) and 692 nm light. Animals 57-64 are treated with systemic injections of SnEt.sub.2 (0.5 mg/kg to 5 mg/kg) and 700 nm light. Incisions sites not exposed with light serve as controls. Dark toxicity controls are performed for each photosensitizer.

DETD **Photodynamic** Inactivation of Extracellular Growth Factors in Wound Healing

DETD For studies on **photodynamic** therapy for abdominal adhesions, a third model, the rat model described by Langer et al. (J. Surg. Res. 59: 344-348 (1995)), is employed. In that model, rats develop intra-abdominal adhesions that can be graded from grade 1 (thin easily separable) to grade 3 (extensive dense tissue masses) (Elkins et al., Fertil. Steril. 41: 926-928 (1984). Intra-abdominal **photodynamic** therapy is performed using methods described by Molpus et al. (Cancer Res. 56:1075-1082 (1996)) for treatment of experimental ovarian cancer. Photosensitizers or conjugates are injected intraperitoneally, followed by administration of red light. The red light is administered through an optical fiber that penetrates the abdominal wall and into a peritoneal cavity, which has been filled with intralipid as a light diffuser. To test whether intra-abdominal PDT can positively affect the degree of adhesion formation, four variables are investigated: (1) light dose, (2) conjugate dose, (3) time between cecal injury and the treatment, and (4) time between i.p injection of conjugate and the delivery of i.p. light. These parameters are varied systematically. Rats are sacrificed four weeks after treatment, and their adhesions are graded in a blind experimental design. Samples of the adhesion tissue are removed for histological staining.

CLM What is claimed is:

1. A method for inhibiting fibrosis in the healing of a wound in a mammal, comprising: (a) administering to a mammal that has an unhealed or partially-healed wound an effective amount of a photosensitizer targeted to macrophages or myofibroblasts by conjugation of a targeting moiety; (b) waiting for a time period wherein said photosensitizer reaches an effective tissue concentration at the wound site; (c) photoactivating said photosensitizer at said wound site by delivering specifically to said wound site light of an effective wavelength and

intensity, for an effective length of time, wherein the dose of **photodynamic** therapy is sufficient to kill from about 10% to about 90% of the targeted cells exposed to photoactivating light, thereby inhibiting fibrosis in said healing of said wound in said mammal.

ACCESSION NUMBER: 1999:69275 USPATFULL
TITLE: Inhibition of fibrosis by **photodynamic** therapy
INVENTOR(S): Trauner, Kenneth, Boston, MA, United States
Hasan, Tayyaba, Arlington, MA, United States
Hamblin, Michael R., Revere, MA, United States
PATENT ASSIGNEE(S): The General Hospital Corporation, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5913884		19990622	
APPLICATION INFO.:	US 1996-741816		19961031	(8)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-26315P	19960919 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Tsang, Cecilia J.
ASSISTANT EXAMINER: Delaney, Patrick R.
LEGAL REPRESENTATIVE: Fish & Richardson P.C.
NUMBER OF CLAIMS: 14
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Figure(s); 1 Drawing Page(s)
LINE COUNT: 695

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[1] : Webster's Revised Unabridged Dictionary (1913)

Infundibulum \In`fun*dib"u*lum\, n.; pl. L. **Infundibula**, E. **Infundibulums**. [L., a funnel, from infundere to pour in or into. See **Infuse**.]

1. (Anat.) A funnel-shaped or dilated organ or part; as, the infundibulum of the brain, a hollow, conical process, connecting the floor of the third ventricle with the pituitary body; the infundibula of the lungs, the enlarged terminations of the bronchial tubes.

2. (Zo["o]l.)
   (a) A central cavity in the Ctenophora, into which the gastric sac leads.
   (b) The siphon of Cephalopoda. See **Cephalopoda**.

See also: [Infundibula] [Infundibulums] [Infuse] [Cephalopoda]

[2] : WordNet (r) 1.7

infundibulum
n : any of various funnel-shaped parts of the body (but especially the hypophyseal stalk)

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